Two Cases of a Dural Arteriovenous Fistula Mimicking a Brain Tumor

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Summary

Dural arteriovenous fistula (d-AVF) is relatively rare. Some cases of atypical locations are often difficult to distinguish from other vascular disorders or tumors because those d-AVFs show various onsets, such as subcortical bleeding and venous infarctions. We encountered two cases of d-AVF with severe brain edema that took adequate time to distinguish from brain tumors. A 68-year-old man visited his local physician complaining of dizziness. He was diagnosed with a cerebral infarction due to the presence of an abnormal cerebellar signal on magnetic resonance imaging (MRI) and was treated by drip infusion. However, he did not recover and was admitted to our hospital with suspicion of a brain tumor. A 75-year-old woman with an onset of progressive dementia and gait disturbance showed severe edema of the right-front temporal lobe on MRI. Both these cases were examined by single photon emission computed tomography or positron emission tomography and were scheduled for craniotomy and biopsy based on the diagnosis of brain tumor. We performed preoperative angiography and found d-AVFs. We embolized the d-AVFs with liquid material and both patients recovered well.

Brain edema from d-AVF or a tumor can be distinguished by carefully reading the MRI with findings such as the distribution of the edemas, differences on diffusion-weighted images, and contrast-enhanced images. Therefore, it is important to provide initial accurate diagnoses to prevent patient mistrust and irreversible disease conditions.

Introduction

Dural arteriovenous fistulas (d-AVFs) are difficult to diagnose because they are rare and show various onsets such as hemorrhage, seizures and venous infarctions. We encountered two patients with d-AVF that took adequate time to diagnose because they appeared to be cerebral tumors with severe cerebral edema.

Representative Cases

Case 1: A 68-year-old man

History of the disease: The patient had vertigo and visited his local physician who diagnosed a cerebral infarction from an abnormal lesion in the right cerebellar hemisphere. Despite drip infusion, his condition did not improve and he was admitted to our hospital with suspicion of a cerebral tumor.

Neuroradiological findings: The brain T2-weighted (T2WI) MRI images showed a large hyperintense area in the right cerebellar hemisphere (Figure 1A). Compared to the large lesion, the mass effect was slight, leading to only slight deformation of the fourth ventricle. The diffusion-weighted images (DWI) did not demonstrate hyperintensity of the lesion (Figure 1B), whereas an apparent diffusion coefficient (ADC) map did show the hyperintensity (Figure 1C). The contrast-enhanced T1-weighted images (CE T1WI) demonstrated right cerebellar cortical enhancement and congested veins (Figure 1D). Abnormal uptake in the lesion was observed by gallium scintigraphy, but not by thallium scintigraphy. We performed cere-

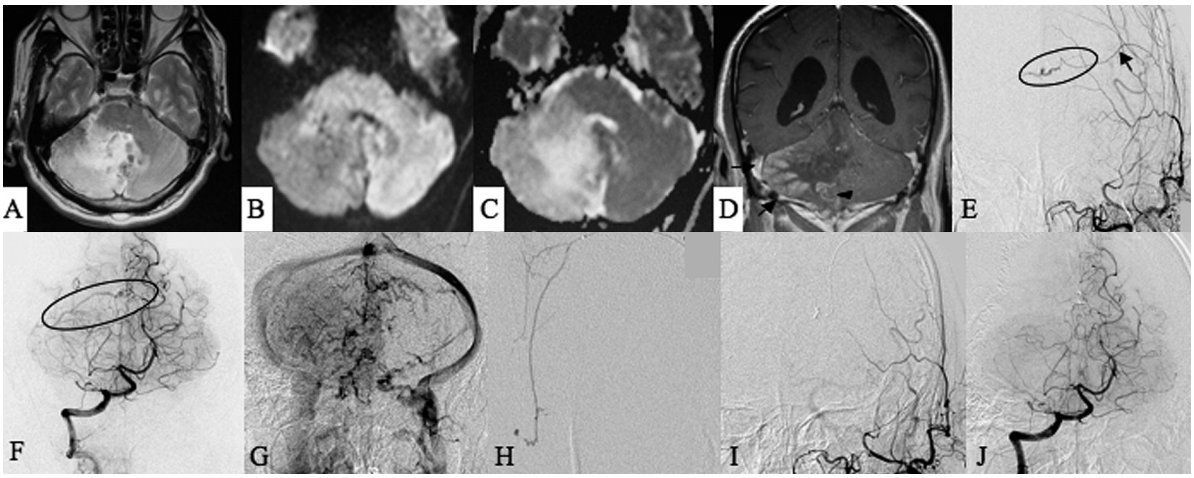

Figure 1 A) T2WI demonstrated high intensity signals in the right cerebellar hemisphere. The mass effect was slight, and the fourth ventricle did not change much in shape due to the large size of the lesion. B) DWI of the same lesion did not show high intensity. C) An ADC map demonstrated high intensity. D) CE T1WI coronal view showed enhancement along the cortex (arrows) and a congested vein (arrowhead). E) A left-external carotid artery angiogram (ECAG) shows a d-AVF (circle) supplied by the left-middle meningeal artery, MMA (arrow). F) Right-vertebral artery angiogram (VAG). The d-AVF (circle) was also fed by the posterior meningeal artery. G) Severe congestion was seen in the cortical veins. H) NBCA was injected from the left MMA. I,J) Postoperative angiogram did not show the d-AVF.

bral angiography and found a d-AVF in which the main blood flows from the left-middle meningeal artery (MMA) and posterior meningeal artery (Figure 1E-G).

Operation (March 2007): We selected transarterial embolization because the shunt point was narrow and not connected to the venous sinuses. A Baltacci 1.8F (Balt) catheter reached the left MMA, and we embolized the shunt point with an injection of 20% n-butyl-cyanoacrylate (NBCA) (Figure 1 H-J).

The patient improved and was discharged from the hospital, but the abnormal findings persisted (Figure 2A). We performed cerebral angiography and confirmed the recurrence of d-AVF in which the main blood flows from the Davidoff-Schechter artery that branch from the left-posterior cerebral artery (Figure 2B).

Second operation (February 2008): A Magic 1.5F MP (Balt) catheter reached the Davidoff-Schechter artery (Figure 2C). We injected 25% NBCA and completely occluded the d-AVF (Figure 2D). Despite the disappearance of the d-AVF, the hyperintensity in the right cerebellar hemisphere remained on the ADC map and T2WI three months after the second operation (Figure 2E,F).

Case 2: A 75-year-old woman

History of the disease: The patient visited her local physician complaining of progressive disorientation and a gait disturbance, and was admitted to our hospital with suspicion of a cerebral tumor.

Neuroradiological findings: The T2WI MRI showed diffuse hyperintense signals in the right-front temporal lobe (Figure 3A). The edema was intractable despite the administration of a large dose of steroid, resulting in bleeding complications. The DWI did not demonstrate hyperintensity of the lesion (Figure 3B), whereas the ADC map did show hyperintensity (Figure 3C). CE T1WI demonstrated cortical enhancement (Figure 3D). We suspected a cerebral tumor because 2-deoxy-2-[18F] fluoro-D-glucose (18F-FDG) PET showed partial uptake (Figure 3E). We performed cerebral angiography despite planning for craniotomy, and found d-AVF in which the main blood flow was from the right-accessory meningeal and the right-ascending pharyngeal arteries (Figure 3F).

Operation (October 2007): Baltacci 1.5F and Ultraflow 1.5F (MTI) catheters reached the right-accessory meningeal artery and the right-ascending pharyngeal artery, respectively. We injected 33% NBCA and 25% NBCA, respectively, into each vessel (Figure 3G), resulting in a complete occlusion of the d-AVF (Figure 3H).

After four months, we confirmed recovery from her symptoms and the disappearance of the d-AVF. The abnormal signal on MRI improved gradually in the ADC map as well as

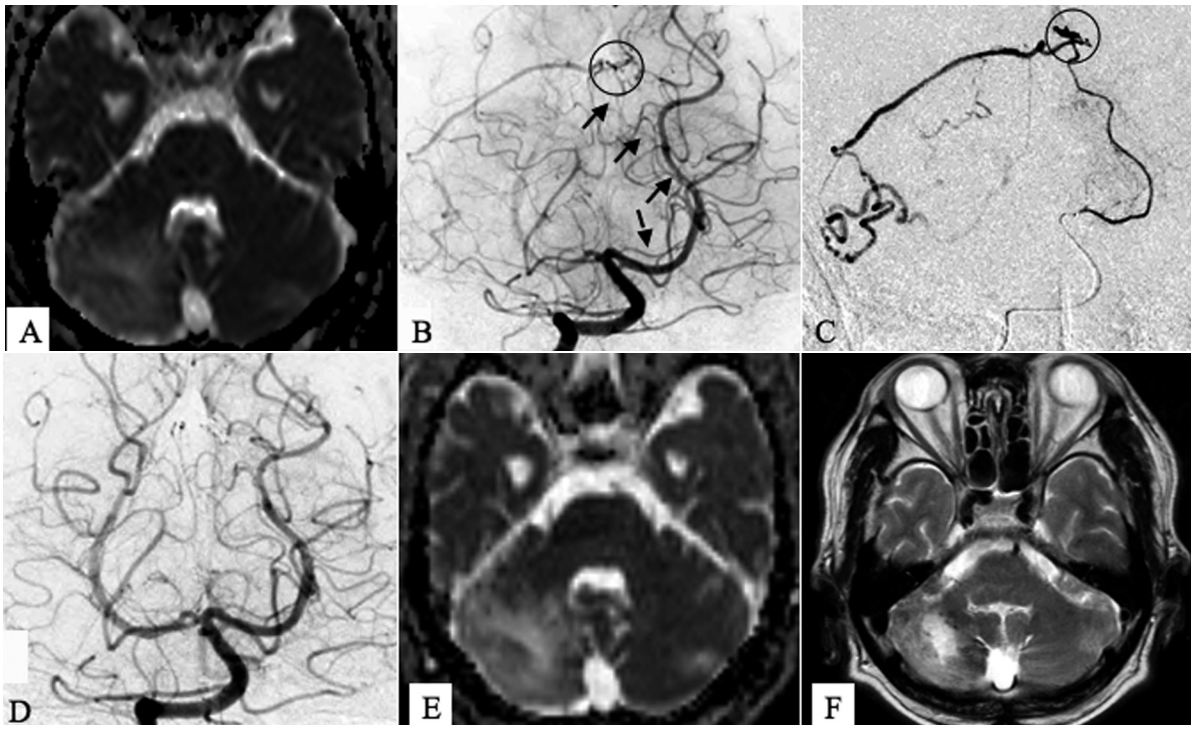

Figure 2 A) The high intensity on the ADC map continued after the first operation. B) Right-vertebral artery angiogram (VAG) showed the recurrence of d-AVF (shunt point, circle) fed by the Davidoff-Schechter artery (arrows). C) A selective Davidoff-Schechter artery angiogram clearly identified the shunt point (circle). D) After injecting NBCA, d-AVF disappeared. E,F) The high intensity on the ADC map and T2WI has remained ever since.

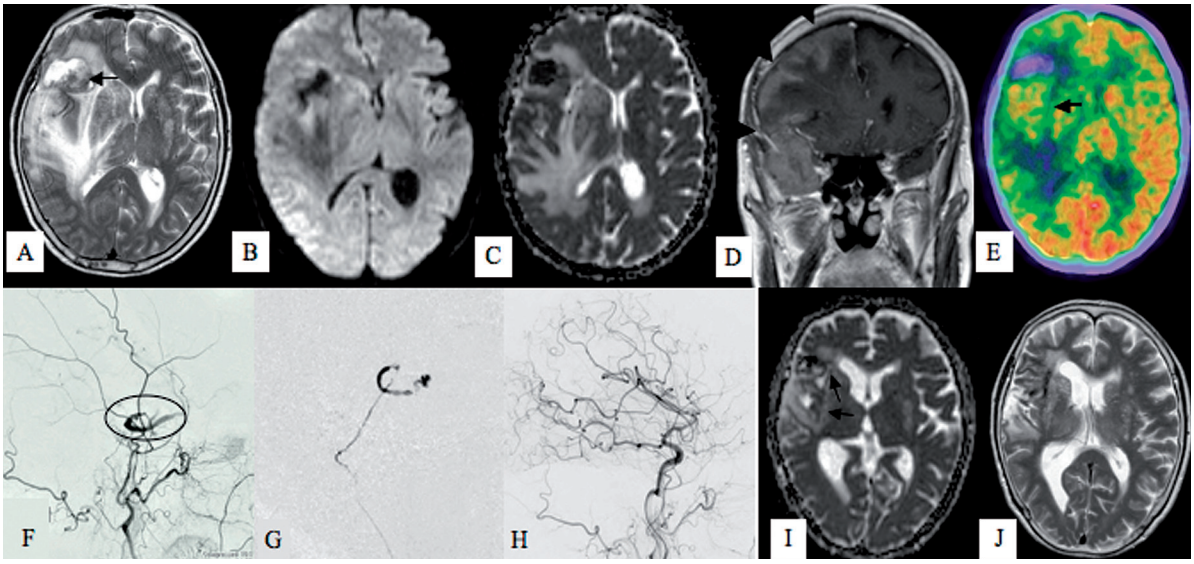

Figure 3 A) T2WI demonstrated a large high-intensity area and a small bleeding area (arrow) on the right-front temporal lobe. B) DWI did not show the high intensity of the lesion. C) An ADC map did show the high intensity of the lesion. D) CE T1WI coronal view demonstrated enhancement along the cortex (arrowheads). E) 18F-FDG PET showed a partial uptake lesion in the right-front temporal lobe (arrow). F) Right-external carotid artery angiogram (ECAG) showed a d-AVF (circle) that was fed by the right-accessory middle meningeal artery (MMA) and ascending pharyngeal artery. G) After injecting NBCA into ascending pharyngeal artery, we embolized the shunt and the proximal part of the sylvian vein. H) D-AVF not seen. I, J) The high intensity seen on the ADC map improved earlier than on the T2WI. However, the high intensity area (arrows), which had not improved on the ADC map, remained on T2WI six months later.

T2WI, but the rate of improvement was faster in the ADC map compared to T2WI. The T2WI hyperintense signal, which was shown on the ADC map, remained hyperintense after six months (Figure 3I, J).

Discussion

D-AVF are relatively rare and most are located at the transverse sinus/sigmoid sinus or the cavernous sinus. At onset, patients complain of headaches, ocular symptoms, and tinnitus. Some of the d-AVFs in atypical locations are often difficult to distinguish from other vascular disorders or tumors because they show various onsets with bleeding, seizures, and venous infarction.

The conditions of the two patients described here were confused with cerebral tumors and severe cerebral edema. We might have been able to diagnose them as venous infarctions or flow voids with MRI findings, but the definitive diagnosis was dependant on cerebral angiography. At the acute phase, venous infarctions, which mainly consist of interstitial edema, do not demonstrate hyperintense signals on DWI, hence, this finding was useful to distinguish them from arterial infarctions. Edema, small bleeding areas, and contrast enhancement of the cortex are also characteristic of venous infarctions [5]. Therefore, careful diagnoses is essential because some venous infarctions occasionally show a ring-like enhancement that mimics a tumor [1], or flow voids as collateral anastomoses develop. Cerebral infarctions or bleeding, leptomeningeal or medullary vascular enhancements, and the ADC map are useful to estimate the prognosis of d-AVF [2,3]. An d-AVF on an atypical location, without communication to sinuses, and draining into cortical veins should belong in Borden's classification type III. Moroever, all are likely to demonstrate leptomeningeal or medullary vascular enhancements on CE MRI. An ADC map is more useful than DWI because it shows vasculogenic edema as a hyperintense signal and interstitial edema as a hypointense signal, whereas DWI shows them as various intensity signals. In case 1, the recurrence was found due to the hyperintense signal that remained on the ADC map. In case 2, the improvement of the hyperintense signal was identified earlier with the ADC map than with T2WI, and thus, was very useful to form the prognosis. Although MR spectroscopy, single photon emission computed tomography, and positron emitted tomography are useful to assist with diagnoses, false positives can occur among the various types and grades of tumors [4]. Lesions with slight edema, such as case 1, are difficult to distinguish from low grade gliomas. Bleeding lesions, blood-brain barrier ruptures, and an increase in partial cerebral blood flow, such as case 2, are difficult to distinguish from malignant tumors.

We occasionally encounter cases with abnormal MRI signals during our daily work, but they may receive inappropriate treatment, observation, or presentation to other hospitals without precise diagnoses. Medical specialization has developed according to advanced technology and if we make an error during initial diagnosis, the patient is at a significant disadvantage. Therefore, accurate diagnosis during initial examination is essential to prevent patient mistrust and irreversible disease conditions.

Conclusions

We introduced two cases of d-AVF that mimicked tumors with severe edema and took adequate time to diagnose. MRI was very useful for the diagnosis and formation of a prognosis for these patients with d-AVF.

References

1 Bakshi R, Lindsay BD et Al: Cerebral venous infarctions presenting as enhancing space-occupying lesions: MRI findings. Journal of Neuroimaging 8(4): 210-215, 1998.
2 Inoue T, Takahashi T et Al: Congestive myelopathy due to cervical perimedullary arteriovenous fistula evaluated by apparent diffusion coefficient values -case report. Neurol Med Chir Tokyo 46: 559-562, 2006.
3 Kwon BJ, Han MH et Al: MR Imaging Findings of Intracranial Dural Arteriovenous Fistulas: Relations with Venous Drainage Patterns. Am J Neuroradiol 26: 2500-2507, 2005.
4 Ogawa T, Tanabe Y et Al: Clinical usefulness of SPECT and PET for diagnosis of cerebral gliomas and radiation necrosis. Jpn J Clin Radiol 52: 792-798, 2007.
5 Tomura N: Computed tomography and magnetic resonance imaging in cerebral venous thrombosis. Journal of Adult Disease 36(2): 143-146, 2006.

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